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**THE ROLE OF THE VESTIBULAR APPARATUS
UNDER WATER AND HIGH PRESSURE**

Robert S. Kennedy

**Naval Medical Research Institute
Bethesda, Maryland**

March 1973

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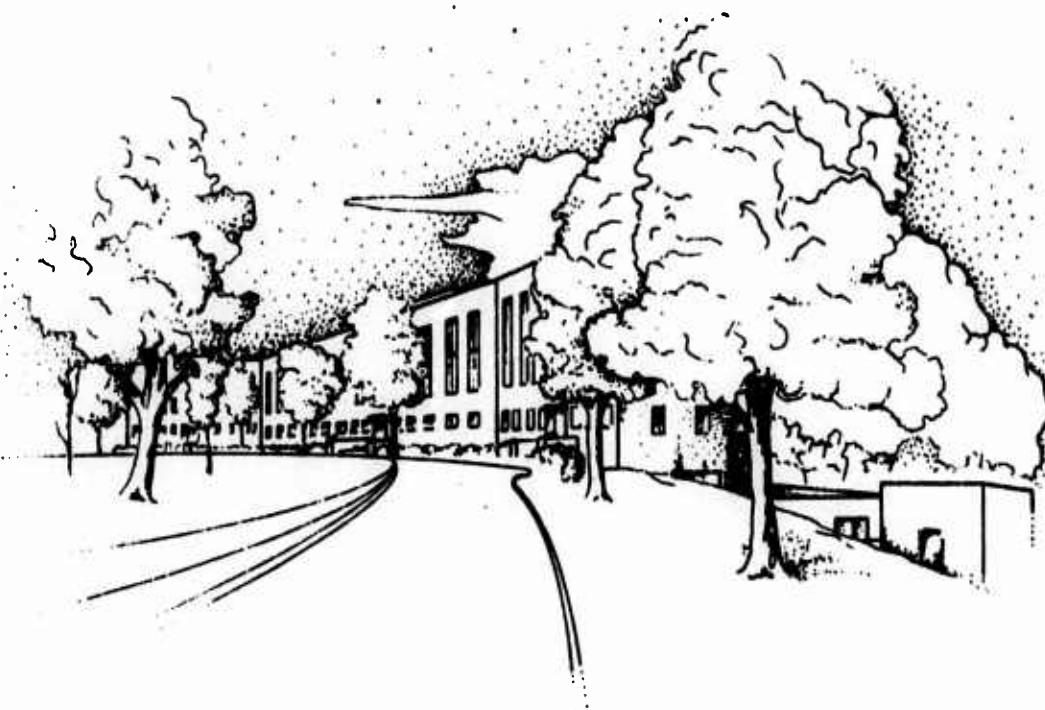
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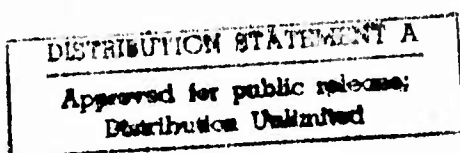
THE ROLE OF THE VESTIBULAR APPARATUS UNDER WATER AND HIGH PRESSURE

Research Report

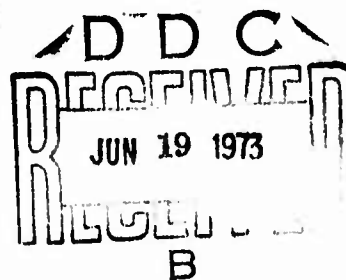
M4306.03.5000BAK9

Report No. 3

MARCH 1973



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DOCUMENT CONTROL DATA - R & D

(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY (Corporate author) NAVAL MEDICAL RESEARCH INSTITUTE NATIONAL NAVAL MEDICAL CENTER BETHESDA, MARYLAND 20014		2a. REPORT SECURITY CLASSIFICATION UNCLASSIFIED	
		2b. GROUP	
3. REPORT TITLE The Role of the Vestibular Apparatus Under Water and High Pressure			
4. DESCRIPTIVE NOTES (Type of report and inclusive dates) MEDICAL RESEARCH PROGRESS REPORT			
5. AUTHOR(S) (First name, middle initial, last name) Robert S. Kennedy, LCDR, MSC, USN			
6. REPORT DATE March 1973		7a. TOTAL NO. OF PAGES 28 54	7b. NO. OF REFS 126
8a. CONTRACT OR GRANT NO.		9a. ORIGINATOR'S REPORT NUMBER(S) M4306.03.5000BAK9 Report #3	
b. PROJECT NO.		9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report)	
c.			
d.			
10. DISTRIBUTION STATEMENT This document has been approved for public release and sale; its distribution is unlimited.			
11. SUPPLEMENTARY NOTES		12. SPONSORING MILITARY ACTIVITY BUREAU OF MEDICINE AND SURGERY (NAVY) Washington, D. C. 20372	
13. ABSTRACT This report briefly reviews literature relating to vestibular functions in underwater medicine, and introduces a more complete reference list, "A Bibliography of the Role of the Vestibular Apparatus Under Water and Pressure: Content-Oriented and Annotated." The author states that the role of the vestibular system in compressed air work is presently underestimated. This review and the broader Bibliography call attention to the incidence of vestibular involvement in compressed air work and provides reference to the background material essential for understanding and future study of vestibular problems.			

UNCLASSIFIED

Security Classification

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
1. Diving						
2. Compressed Air Work						
3. Bibliography: Content-Oriented, Annotated						
4. Vestibular Function						
5. Vestibular Symptoms						
6. Diving Medicine						
7. Compressed Air Sickness						
8. Decompression Sickness						
9. Vestibular Hits						
10. Diving History						
11. Vestibular Diagnosis						
12. Vestibular Pathology						
13. Caisson Disease						

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ABSTRACT

This report briefly reviews literature relating to vestibular functions in underwater medicine, and introduces a more complete reference list, "A Bibliography of the Role of the Vestibular Apparatus Under Water and Pressure: Content-Oriented and Annotated." The author states that the role of the vestibular system in compressed air work is presently underestimated. This review and the broader Bibliography call attention to the incidence of vestibular involvement in compressed air work and provides reference to the background material essential for understanding and future study of vestibular problems.

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Compressed Air Work

Bibliography: Content-Oriented, Annotated

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PROLOGUE

Large research efforts have been mounted to study better the relationships of vestibular functions to aerospace systems, although similar efforts have not been undertaken in underwater medicine. It is felt that a greater requirement for research into vestibular implications exists in underwater medicine than in aviation medicine.

The present report is a literature review; very little information was obtained from personal observation. However, because in the recent past such small emphasis has been placed on this area of investigation, it appeared that an evaluative integration of the research literature, with many speculations, would be more heuristic than a mere summary of the facts. The plan of this report therefore is to present briefly a portion of what is considered relevant and to provide a more complete reference list elsewhere (Kennedy, 1972).

BACKGROUND

Snell (1896) attempted to document "...cases of auditory vertigo ...because they have not previously been met with and recognized by writers on the subject [p. 74]." Yet earlier reporters of compressed air work also had recognized such a malady. For example: (a) Curnow (1894) published an article in Lancet two years earlier than Snell with the term "auditory vertigo" in the title; and (b) Smith (1873), twenty years earlier, claimed that "Affections of the ears are mentioned by

*From Bureau of Medicine and Surgery, Navy Department, Research Subtask M4306.03.5000BAK9. The opinions and statements contained herein are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

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Presented at Fifth Symposium on Underwater Physiology, 21-25 August, 1972, Freeport, British Bahamas.

every writer on the subject of compressed air and are extremely common [p. 25]", further, "vomiting" [p. 26], "vertigo" [p. 27], and "dizziness" [p. 29] were also described by Smith before Snell as symptoms of "Caisson disease" [p. 25], a term both authors feel they coined; and (c) many of these same symptoms were also mentioned by Bert in his famous work (1878), and by van Rensselaer (1891).

A lack of awareness of previous workers' reports regarding vestibular symptomatology as part of a constellation of symptoms which occur with some frequency in hyperbaria is not limited to Snell, but occurs often in the history of compressed air work.*

There are several reasons why the full importance of vestibular problems may not have been realized: (a) many articles of merit were written in German, Italian, and French, although a major portion of the decompression sickness literature is in English; (b) of the foreign articles cited in the diving medicine "Sourcebooks" (Hoff, 1948; Greenbaum & Hoff, 1954; Greenbaum & Hoff, 1966), English titles were provided only for those studies which were written in Russian; (c) specific pertinent categories (e.g., "ear lesions"), which occurred in Volume I of the Sourcebook (Hoff, 1948) were subsequently dropped; (d) pain-type symptoms (type I - Griffiths, 1969) are more clear-cut, more common, and are usually a cause for self-referral, whereas the various vestibular-type symptoms (e.g., vertigo) can be due to several causes in compressed air work (e.g., syncope, migraine, oxygen toxicity) and are less tangible

*It should be noted that Shilling (1938, 1941a, b) suggested that after joint problems, otological problems were next in importance for future study, but this suggestion does not appear to have been taken up by workers in the field.

than pain; (f) physicians connected with underwater medicine are more likely to be specialists in internal medicine or neurology than in otolaryngology.

Because the Navy Diver's Manual (1970) and other guides mention the role of the vestibular apparatus very little, this review was undertaken.

An Annotated Bibliography (Kennedy, 1972) of studies concerning the roles of the vestibular apparatus in compressed air work has been published and is available. The Bibliography is organized into six categories and the studies are sorted and listed within appropriate categories. Generally, the studies are annotated and some cross-referencing exists. About 1,000 references are included.

The categories are:

1. Vestibular symptomatology reported in connection with compressed air work.
2. Clinical diagnosis of vestibular symptoms with relevance to hyperbaria.
3. Provocative tests of the positive function of the vestibular apparatus to be used as pre-post measurements of normal functions.
4. Illusory phenomena occasioned by water and similar environments which may involve or interact with vestibular functions.
5. The potential use of the vestibular system as a navigation aid under water.
6. Relevant auditory studies.

The purpose of the present report is to summarize and evaluate the main findings of the studies listed under "Vestibular symptomatology

reported in connection with compressed air work." Citations in the other categories are in the main bibliography.

METHOD

The chief criterion employed by which an article was placed into this category was that vestibular symptomatology was cited by an author in connection with the exposure of humans (usually) under water or pressure; however, other considerations were also used. For example, not all articles which dealt with aerotitis media were included, since so many exist and since the mechanism appears to be reasonably well understood. However, an attempt was made to include all the literature concerning alternobaric vertigo (e.g., Lundgren, 1965) the high pressure nervous system syndrome (e.g., Bennett & Towse, 1971), et cetera. Some references which it is felt contain useful methodologies for future study of this problem were also included (e.g., Flanagan, 1949).

Generally, original sources were referenced, although occasionally articles which reviewed the work of others were included. Other criteria were employed and are described in more detail elsewhere in the Annotated Bibliography. For the most part, the literature was assembled by searching the reference lists of modern authors and proceeding backwards. Particular attention was paid to articles which were not written in English. The three "Sourcebooks" (Hoff, 1948; Greenbaum & Hoff, 1954; 1966) and Shilling & Werts (1971) were then consulted for articles which may have been missed by this approach. In addition, the general otolaryngologic literature was surveyed.

RESULTS AND DISCUSSION

The number of studies which report vestibular symptoms in hyperbaria is presently increasing, although about as many appeared before 1900 as between 1900 and 1940. From the literature review (Kennedy, 1972) it seems that the number of studies approximately doubles each decade since 1900, and presently, the number of articles is probably growing at an equivalent or faster rate than the scientific literature in general. The three "Sourcebooks" (Hoff, 1948; Greenbaum & Hoff, 1954; Greenbaum & Hoff, 1966) and Shilling & Werts (1971) show a slower growth rate than this for compressed air work studies in general. For example, studies which mention vestibular-type symptoms are infrequent between 1940-1955, a period within which the "Sourcebooks" indicate that diving medicine investigation grew at the most rapid rate before or since (about 4,000 citations in the six-year period of January, 1946 to December, 1951).

Approximately 300 studies appear in Section I of the larger Bibliography (Kennedy, 1972), which report vestibular symptoms in compressed air work. The reported incidence of vestibular symptoms in these studies ranges from zero (i.e., none reported) (e.g., Bond, 1966; Paton & Walder, 1954), to 28% (Hill, 1912, reviewing Heller, et al., 1900), to 40% where a very liberal criterion was employed (Kennedy & Diachenko, in preparation) to more than 50% with "ear problems" (87 out of 161) in tunnel workers (Bassoe, 1913, p. 541). In the latter study a further breakdown showed "33 complained of dizziness..., 6 of vomiting..., 6 had blind staggers, that is, labyrinthine vertigo, with nystagmus [p. 527]."

Therefore, 28% probably involved the vestibular system itself (cf., Hill, 1912, above). In modern saturation diving studies, as many as 50% or more of the subjects have been reported to have experienced vestibular-type symptoms (e.g., "dizziness" and "nausea" [p. 1154] were complaints made by both subjects [Bennett & Towse, 1971]). In addition, related CNS (i.e., type II*, Griffiths, 1969) symptoms also appear to be occurring with greater frequency in saturation diving unless special precautions are taken. In these saturation diving studies the group sizes are typically small--generally 2 to 4 persons (Bühlmann, Matthys, Overrath, Bennett, Elliott, & Gray, 1970; Hamilton, MacInnis, Noble, & Schreiner, 1966; Sundmaker, 1972). However, in a report of 83 saturation diving accidents (11-23 ata), 13% involved the labyrinth (Bühlmann & Waldvogel, 1967) and nearly all of these (9 out of 11) required treatment; whereas, of the remaining (non-labyrinthine) accidents, a smaller proportion (49 out of 71) required treatment.

Vestibular symptoms in divers have been mentioned in pressure chambers and open-sea dives for various working conditions, gas mixtures, and depths (Rubenstein & Summitt, 1971). They have also been reported in submarines (Uffenorde, 1948), caissons (Hill, 1912), and following breath-hold dives (Pauley, 1965). They have occurred during compression (Shilling, 1937, p. 379), and just after (Rivera, 1963) or long after (Keays, 1909) decompression, and also under isobaric conditions (Sundmaker, 1972). They appear to occur also in guinea pigs and squirrel monkeys (McCormick, Higgins, Clayton, & Brauer, 1971) under high pressure.

*As opposed to type I (i.e., pain).

Vestibular symptomatology (viz., "nausea and vertigo," Chouteau, Ocana de Sentuary, & Pironty, 1971) are among the symptoms mentioned in connection with the high pressure nervous system syndrome (HPNSS) (Overrath, Matthys, & Buhlmann, 1970; Bennett & Towse, 1971).

Residual vestibular defects have been reported both in divers (Lehmann, Held, & Werner, 1970; Plante-Langchamp, Maestracci, & Nicolai-Harter, 1970; Kennedy & Fregly, in preparation), and caisson workers (Lestienne, 1933; Rozsahegyi, 1959). Furthermore, Bertoin (1953) feels these labyrinthine symptoms specifically worsen with time. Residual central nervous system deficits (EEG abnormality) were high (50%) in a group which experienced labyrinthine symptoms of decompression sickness (DCS) (Rozsahegyi & Roth, 1966a, b), although true control groups were not shown in that study.

In terms of the number of instances of vestibular-type involvement in DCS, probably the best data are found in Rivera (1963), where out of 935* cases "dizziness or vertigo" were reported 80 times; "nausea or vomiting," 74 times; "visual disturbances," 64 times; "incoordination," 9 times; "equilibrium disturbances," 7 times; and "auditory disturbance," 3 times. Although often these symptoms occurred along with others (notably localized pain), still dizziness, nausea, or visual disturbance was a premonitory sign in 5% of all the cases.

Studies of a history of vertigo only (as opposed to vestibular symptoms in general) in divers have shown that a high proportion of

*For an earlier, larger, but less delineated review (i.e., 1,361,461 decompressions) Behnke (1951) should be consulted.

divers, i.e., between 12% to 40%, (Lundgren, 1965; Terry & Denison, 1966. Vorosmarti & Bradley, 1970) have experienced vertigo at least once in their careers.

Decompression sickness (mainly, type II) was responsible for 10% of civilian diving deaths, yet almost half the deaths (30) were cause unknown (Bayliss, 1969). Possibly disorientation complicated by DCS could have been a factor in these 30.

In tests at pressure, although the vestibular-ocular reflex did not change with increasing depth (Adolfson & Fluor, 1967), postural disequilibrium (body sway) did increase as pressure increased (Adolfson, Goldberg, & Berghage, 1972). It is known that noise is an adequate stimulus for the vestibular system (Ades, Graybiel, Morrill, Tolhurst, & Niven, 1957; Parker, von Gierke, & Reschke, 1968) and the "Tullio (1925) effect" suggests that noise can produce body sway. It has been shown that pressure chambers are noisy (Summitt & Reimers, 1971) and this factor should be considered in future studies.

One author (Reuter, 1971) feels that "...90% of the medical problems of the sport diver are centered around the middle ear [p. 3]."

From these results it appears that vestibular type symptoms are prominent in compressed air work, yet the actual incidence may be much larger than is reported here and several reasons are offered for this belief:

1. In compressed gas work dizziness, nausea, vertigo and vomiting, and occasionally ataxia, are considered as vestibular symptoms, although only vertigo is discussed. However, other symptoms also occur

from stimulation to the vestibular system. These include drowsiness, pallor, sweating, and salivation, as well as various sorts of visual phenomena (e.g., nystagmus, apparent movement). If a liberal criterion is employed when analyzing U. S. Navy diving accident records, as many as 40% of all DCS accidents (oxygen toxicity and compression accidents omitted) contain central nervous systems (CNS) symptoms which may indicate vestibular involvement (Kennedy & Diachenko, 1972), although 15% is a more realistic figure (cf., Rivera, 1963). As shown also by Rubenstein & Summit (1971) in their study of vestibular derangement, vestibular involvement appears to be on the increase. This increase may be connected with saturation diving, and because of the long periods spent under pressure by caisson workers, there may be a connection between them. Perhaps much is to be learned by reviewing the older literature of caisson work.

2. It is generally agreed that CNS-type symptoms occur sooner after decompression than pain-type symptoms. If so, it is possible that vertigo could precede experiences of localized pain and, (a) be ignored, or (b) self-limited; either by keeping the head still (cf., Bennett, 1970, where this worked while at pressure), or by sleep.* Pain could develop later. Therefore, a physician could see a patient who presents with pain symptoms but who may have also had vestibular symptoms previously. These vestibular symptoms may be missed in the course of treating the former. Additionally, vertigo or dizziness may not be

*Alcoholic intoxication could potentiate the vestibular symptoms (Bergstedt, 1961) and/or enhance sleep.

considered mainly by the respondents (e.g., Navy divers) and are not reported as frequently as they occur for this reason.

3. Diver accident records used by the U. S. Navy contain physically a broad space in which to record pain-type symptoms and the physician is encouraged by the layout of the form to add descriptive comments for pain on the form beside the time it occurred. However, he is enjoined from doing this for vestibular-type symptoms since the spaces beside dizziness, vertigo, et cetera, are blackened out.

4. There may be a tendency to consider type I and type II symptoms of DCS as mutually exclusive categories (see McCallum, 1968, for a review of DCS studies reported between 1914-1966). Thus, if a person reported severe pain and mild dizziness there might be a tendency to classify this as type I. Further, the tangibility of the pain-type symptoms, with probably higher cure rate, may also cause them to be favored as a diagnostic category.

5. Provocative tests of Eustachian tube clearing at 50 psi are conducted prior to Navy diver training (Shilling & Everley, 1942), but not necessarily prior to civilian Scuba training. If Eustachian tube patency is negatively related to a susceptibility to vertigo, then, other things being equal, data from Navy diver records may underestimate the problem when generalized to include the potential incidence of vestibular problems in all diving.

6. Because a form of apparent movement is experienced less by alcoholics (Voth, 1965), and because a "fullness of habit" (van Rensselaer, 1891, and others) is common in compressed air workers,

then perhaps experiences of vertigo in career divers may be less in these groups than in sport divers.*

7. Sometimes vestibular-type symptoms are not listed in reports of decompression sickness (cf., Paton & Walder, 1954, p. 10). One must assume that either too-few-to-mention were obtained, or they were missed on the patient's interview because the response category (e.g., dizziness, vertigo, et cetera) was not on the physician's form.

8. The qualitative coding of U. S. Navy DCS symptoms employs higher numerical numbers for vestibular-type symptoms (e.g., "12" and "20" for "dizziness/vertigo" and "equilibrium disturbances" [Doll & Berghage, 1967, p. 29]) and these symptoms tend not to be grouped together. Computer sorts, performed serially, could underestimate the incidence of these symptoms if they occur along with other symptoms with lower code numbers, since the computer cards would need to be replaced to check on the incidence of these symptoms after they had appeared in other categories.

9. Nystagmus (spontaneous and otherwise) has been mentioned directly (Hoche, 1897; Sundmaker, 1972) and indirectly (Bert, 1898, pp. 381-385). In addition, disconjugate eye movements in decompression have also been mentioned (Erde, 1963). These eye movement responses are generally not listed as a sign or symptom of DCS in the reviews which have appeared (cf., item 1, p. 8). Yet characteristics of nystagmus (direction, rate, frequency, et cetera) are a useful aid to diagnoses of problems involving vestibular pathways.

*This factor may be a training or natural selection variable since a high fluid exchange rate may be a consequence of "fullness of habit," but a high fluid exchange rate has also been shown to afford some protection from decompression sickness (Warwick, 1942, 1943).

10. Reporting the symptomatology of altitude DCS, Gray, Mahady, Nasland, & Wigodsky (1946) consider "nausea, vomiting, pallor, sweating, faintness [p. 339]" as circulatory reactions, although vestibular stimulation causes similar symptoms.

11. In some studies of DCS (e.g., Golding, Griffiths, Hempleman, Paton, & Walder, 1960) only cases "severe enough for the man to bring himself for treatment are included [p. 168]." This approach should overestimate the incidence of type I (pain) and underestimate type II (CNS) and consequently vestibular involvement, since the latter are generally considered as type II symptoms.

12. Because the incidence of the individual symptoms reported by investigators usually add up to 100% (cf., e.g., Erdman, 1913) this means that accessory symptoms or benign, accompanying symptoms, are often ignored in these reports. Thus there may be a tendency to classify a symptom according to the patient's discomfort rather than other considerations. Pain is tangible and hurts, and type I diagnoses may be made even though mild type II symptoms may also have been present but are not reported or recorded.

13. Although probably pressure vertigo is more common in divers than flyers, reference to this malady was not seen in the U. S. Navy Diving Manual (1970), but it is mentioned in the U. S. Navy Flight Surgeon's Manual (1968).

For these reasons and others, it is felt that greater attention should be paid to vestibular involvement in compressed air work.

CONCLUSIONS AND RECOMMENDATIONS

The results reported above indicate that greater attention should be paid to vestibular involvement in hyperbaria. Future plans and programs should include:

I. An improved nosology for the differential diagnosis of vestibular involvement should be developed. Edmonds (1971) has suggested a useful classification system for vertigo but his system should be broadened to include other forms of vestibular symptomatology (e.g., nystagmus, ataxia, nausea, disorientation, et cetera), which also occur under water and pressure. Speculation about the potential causes of these vestibular symptoms should be made more freely in order to aid others in differential diagnoses, and so that symptoms of vestibular origin can be separated from the same or similar symptoms due to other causes. In compressed air work the mechanisms which could involve the vestibular system and result in vestibular symptomatology are:

1. Bubble formation and lesions which are either:
 - a. Cerebellar (Erdman, 1913; van Rensselaer (1891)
 - b. Medullary nuclei (Roszahegyi, 1959)
 - c. Semicircular canal (Heller, et al., 1900)
 - d. Other "aural lesions" (e.g., nerve VIII, utricle, et cetera) (Shilling & Everby, 1942, p. 669)
 - e. Spinal (Schumcher, 1967)
 - f. Cervical (Jongkees, 1969)
 - g. In areas (floor, ceiling) of the IVth ventricle
2. Caloric irrigation (Stang & Wiener, 1970; Rowe, 1961; Lanphier, 1957); of the external ear by water or gas

3. Temporo mandibular joint problems (Costen, 1934*; Kelly & Langheinz 1946*; Harvey, 1948*; Pinto, 1966) from long-term mouth-piece use
4. Noise (Parker, et al., 1968*; Ades, et al., 1957*) from chambers, et cetera
5. Wax/cerumen in the ears (Fields, 1958) which could result in different caloric, or pressure, stimuli to the ears
6. Exostoses (Bayliss, 1968*)
7. Barotraumatic otitis media (Behnke, 1969; Melville Jones, 1957; King, 1966)
8. Differential pressure of external auditory canal due to a sea of the pinna (Pagano, 1959)
9. Inner ear barotrauma (Edmonds, 1971)⁺ perhaps due to "pressure...because of blocks in endolymphatic circulation" (Rubenstein & Summitt, 1971, p. 291)
10. Cupulolithiasis (Schuknecht, 1969*)[#]

*These references do not specifically connect diving with the vestibular problems which are cited, although it is felt that such connection might exist.

+This may be the same as #1, bubble formation/lesions.

#Aside from compressed air work, whether the otoconia (calcium carbonate concretions) from the utricle can be given off and lodge in the ampullae of the posterior (inferior) canal is not certain but possible (Lim, in press). However, the symptoms reported by Sundmaker (1972) in a deep dive occurred after a long latency following a pressure change. This long latency suggests that a biochemical explanation is more probable than a neural or mechanical one and cupulolithiasis may be an analogous syndrome. (See particularly Lim, in press.) Further, the fact that Radomski and Bennett (1970) showed that increased calcium retention occurred during an exposure to high pressure supports this notion and may also be related.

There are other circumstances or syndromes which have occurred in hyperbaria where vestibular-like symptoms are reported but where it is not certain that the vestibular system is or is not directly involved. These include: (1) migraine (Engel, 1944; Anderson, Heyman, Whalen, & Saltzman, 1965); (2) Valsalva problems (Edmonds, 1971); (3) syncope (Lee, Matthews, & Sharpey, 1954; Langer & Mansure, 1971); (4) oxygen toxicity and related gas mixture problems (cf., Bennett, 1967); (5) the inversion of the stomach contents with respect to gravity when descending or swimming downward (Fields, 1958); (6) cerebral gas embolism (Gillen, 1968); and (7) circulatory problems (Gray, et al., 1946); (8) perceptual problems occasioned by the environment (viz., visual articulation, submersible motion, neutral bouyancy, et cetera [cf., Kennedy, 1970]); (9) sudden deafness which occurs occasionally after deep diving (Harris, 1969), and others.

II. It is felt that a questionnaire should be developed to serve as a controlled interview to document the experiences of divers related to vestibular symptomatology and to define the magnitude of the problem. For leads in such an effort studies by Clark & Nicholson (1954), Flanagan (1949), Graybiel & Clark (1945), Hardacre & Kennedy (1963), Vinacke (1946 a,b,c,d,e) and Pashalian, Crissy, Siegel, & Buckley (1952) should be consulted.

III. A study of the following relationships may be interesting: (a) vestibular DCS (Rozsahegyi, 1959); (b) Meniere's disease (Simpson, 1965); (c) syncope/vagotonia in high pressure and after decompression (Chouteau, et al., 1971; Coles & Knight, 1961; Donnell, 1960);

(d) orthostatic intolerance from water immersion (Graveline, 1961); (e) sodium retention in high pressure environments (Radomski & Bennett, 1970); (f) migraine after decompression (Anderson, Whalen, & Saltzman, 1964); (g) release of ADH after vestibular stimulation (Taylor, Hunter, & Johnson, 1957); and (h) the advantage of a high fluid exchange rate in protection from DCS (Warwick, 1942, 1943) may be connected.

IV. Electroencephalographic changes have been observed in caisson workers who have had vestibular "hits" (Rozsahegyi & Roth, 1966). Because: (a) EEG changes (microsleep) occur in connection with the high pressure nervous system syndrome (Fructus & Fructus, 1971); (b) REM sleep is absent when lesions in the vestibular nuclei have been performed (Pompeiano & Morrison, 1965; Morrison & Pompeiano, 1965); (c) electro-oculograms can influence EEGs (Mulholland & Evans, 1965; Lippold, 1970); and (d) high pressure can influence eye movement responses (Dolatkowski, Torbus, Dega, & Klajman, 1966), these interrelationships should also be studied.

V. The incidence of all forms of DCS is probably about 1% of all exposures (Keays, 1909; Doll, 1965), however, a very low base rate (0.0318%) occurred when three factors were employed to preselect caisson workers: (a) ear drum inspection; (b) ability to equalize (ear drum) pressure; and (c) no untoward effects on the first work shift (Kooperstein & Schuman, 1957). In addition, Kelly & Langheinze (1946) have shown that dental adjustments made surgically can be used to correct acute otitis media. The relationships between the findings of Kooperstein & Schuman (1957) and Kelly & Langheinze (1946) should be explored relative to the

labyrinth and for what aid they may provide in understanding various forms of DCS.

VI. It was shown that while $< 1\%$ of all decompressions result in accidents, $> 15\%$ of all career workers develop aseptic bone necrosis (Alvis, 1972), even though they may not have experienced DCS symptoms per se. Perhaps a similar "iceberg" relationship exists regarding vestibular decompression sickness. The reports by Rozsahegyi in caisson workers suggest that a vestibulo/neurological examination of divers definitely should be undertaken (cf., Walder, 1967) before and after work in compressed air environments.

VII. If the embryology of sensory systems is considered in connection with DCS a better understanding of the mechanisms involved may result. For example, development of chemoreceptors (e.g., vision) and osmoreceptors (e.g., taste and smell) occurs differently from development of mechanoreceptors (vestibular, cutaneous, auditory). The latter develop in embryo from the alar plate which gives rise to what become the vestibular (VIII) nuclei. From these nuclei what ultimately become vestibular, auditory, and cutaneous/proprioceptive systems develop (Simpson, 1965; Snider & Lowy in Graybiel, ed., 1970). In addition, the corpora quadrigemina and the cerebellum are also embryologic outgrowths from the same origin. Perhaps auditory, skin, and vestibular symptomatology of DCS should be considered together because they have similar beginnings. Further, tremor may be a cerebellar (or vestibular or spinal) phenomenon related to the dizziness and nausea cited above, and both could also be related to "niggles" which are observed in connection with the high pressure nervous system syndrome.

EPILOGUE

The vestibular apparatus is phylogenetically and ontogenetically a primitive and early developing sensory system. The system is anatomically lodged in the petrous portion of the temporal bone and vestibular responses are largely automatic and unconscious. Therefore, it is physically, physiologically, and behaviorally probably the least accessible of all the sensory systems. Yet, the evidence is mounting that this system is being implicated more and more in human compressed air work. It is urged strongly that greater attention be paid in the future to the involvement of vestibular functions under water and high pressure.

Other implications not mentioned in this report but cited elsewhere (Kennedy, 1972) include: (1) the use of the vestibular system as a navigation aid, (2) vestibular problems under water of a spatial orientation/disorientation nature, and (3) the utility of baseline vestibular pretesting to determine whether vestibular damage occurs following a career in compressed air work.

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